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Chapter 10

Gastrointestinal Diseases

Now good digestions wait on appetite, and
health on both.
William Shakespeare
Macbeth, III.iv. (1605–1606)

Introduction

The gastrointestinal system extracts nutrient- and energy-yielding molecules from plant and animal foods and digests them to smaller subunits that can be absorbed. Here also, indigestible food wastes are prepared for excretion. The full complement of essential nutrients is required for normal gastrointestinal function, as for any other bodily system, and malnutrition can adversely affect nutrient digestion and absorption. Similarly, infectious and other diseases that impair gastrointestinal function can impair nutritional status. These interrelationships are important in infant malnutrition as well as in the adult conditions reviewed in this chapter.

Historical Perspective

The history of digestive physiology is remarkable both for the early understanding of its importance to nutrition and health and for the imaginative research experiments that elucidated its function. This early history was reviewed by McCollum (1957).

Hippocrates (460–370 B.C.) believed that although there were many different kinds of foods, there was only one aliment. As early as the second century A.D., Galen (130–200) studied digestion in swine and concluded that the function of the stomach was to convert food into particles small enough to be absorbed.

In the 18th century, de Reaumer (1683–1757) investigated digestion in birds. He inserted food into metal tubes closed at the ends by screens and placed the tubes in the stomach of a predatory bird. When the bird regurgitated the tubes, he was able to observe the dissolution of bones and the partial dissolution of meat in a fluid that tasted salty and bitter. Later, he performed similar experiments in dogs. Similar studies were conducted by Stevens in 1777 using as a subject a man who had earned his living for the past 20 years by swallowing stones. Stevens placed foods in perforated silver containers, and the man swallowed them. By the time the containers were recovered in stool, all foods except seeds were observed to be completely dissolved.

Perhaps the best known human digestive experiments were conducted by Beaumont (1785–1853) on a patient, Alexis St. Martin, who had a permanent opening from his stomach to the outside of his body (a fistula) as a result of a gunshot wound. Beaumont had easy access to and from St. Martin's stomach through the fistula and was able to observe gastric action on many types of foods.

Modern digestive physiology began in the 20th century with the identification of digestive enzymes. Although the basic principles of digestive physiology have been known for decades, the complex neuroendocrine interactions between the brain, digestive tract, and other tissues that control and regulate gastrointestinal function are as yet incompletely understood and are the subject of much current investigation (Nicholl, Polak, and Bloom 1985).

Significance for Public Health

Data from several national surveys provide evidence that these gastrointestinal conditions are extremely prevalent and cause considerable impairment of health and functional ability in the American population. In 1984, the incidence of digestive diseases and conditions that required medical attention or restricted normal activities was 7.6 per 100 persons per year. The incidence rate was highest for infants, children, and young adults (10.3 to 11.9 per 100) and lowest for adults ages 45 to 64 (4.7 per 100). Gastrointestinal conditions were responsible for an average of 32 days per 100 persons per year of limited activity among people of all ages, but among adults over age 65, the rate of restricted activity was 63 days per 100 persons per year (NCHS 1986).

Also in 1984, diseases of the digestive system accounted for a total of over 4 million hospitalizations at an average length of stay of 6.3 days. For patients under age 15, the leading gastrointestinal causes of hospitalization were appendicitis, noninfectious gastroenteritis, and colitis. For patients ages 15 to 44, gastroenteritis and colitis were the leading causes. For adults ages 45 to 64, ulcers and gallstones predominated, and for adults over age 65, cancer, diverticula, and gallstones were the most important causes (NCHS 1987b). The total direct medical cost for gastrointestinal diseases in the United States is estimated at $17 billion per year (Klurfeld 1987).

Mouth, pancreas, colon, and rectal cancers were responsible for about 20 percent of all cancer deaths in 1986. There were 245,000 new cases of cancers of the mouth and digestive tract, and these diseases caused 125,000 deaths that year. Cancers of the colon and rectum are the second leading cause of new cancer cases in men and the third leading cause in women (see chapter on cancer). In 1986, 60,000 Americans died of colon and rectal cancers (Silverberg and Lubera 1987).

The interaction between infectious diarrheal diseases and malnutrition is the primary cause of infant and child mortality and morbidity worldwide (see chapter on infections and immunity), accounting for about 1.5 billion diarrheal episodes annually (Chen and Scrimshaw 1983). In the United States, these conditions are relatively rare but still were estimated to be responsible for about 5 percent of all infant deaths in 1985 (Wegman 1986).

Because most persons with diverticular disease are asymptomatic, the true prevalence of this condition is unknown. Diverticular disease is common in industrialized countries but extremely rare among rural populations in developing countries. Its frequency ranges from about 5 to 40 percent of subjects in Westernized nations (Painter 1985). Among Americans surveyed, 8.4 out of every 1,000 persons reported having diverticula (NCHS 1986). Frequency increases with age, and up to 70 percent of people from age 40 to 70 may be affected (Taylor and Duthie 1976). In 1980, diverticulosis caused 200,000 hospitalizations, incurring health care costs that exceeded $300 million dollars (Almy and Howell 1980). For hospitalizations recorded in 1984, the average hospital stay for such disorders was 8 days (NCHS 1987b).

Data from the National Hospital Discharge Survey, conducted by the National Center for Health Statistics, indicate that gallstones were responsible for 488,000 operations annually in the United States; the average hospital stay for this condition was 7.6 days in 1984 (NCHS 1987b). Nearly 3,000 Americans died of complications of gallbladder disease in 1986 (NCHS 1987a).

The estimated incidence of inflammatory bowel diseases increased from 1.9 cases per 100,000 population in the period 1935 to 1954 to 6.6 cases per 100,000 in 1965 to 1975. Projections based on incidence rates suggested that 20,000 to 25,000 new cases of this condition were admitted to hospitals in the United States in 1980 (Kirsner and Shorter 1982). The National Center for Health Statistics reported that 9.6 of every 1,000 Americans surveyed stated that they had enteritis or colitis (NCHS 1986).

Chronic liver disease and cirrhosis are the ninth leading cause of total deaths and the seventh cause of disease in the United States. They caused the death of more than 26,000 Americans in 1986 (NCHS 1987a). These conditions are discussed in more detail in the chapter on alcohol.

Scientific Background: The Digestive System

The primary functions of the digestive system are to ingest, digest, absorb, transport, and excrete food components. It accomplishes these tasks by means of the various digestive organs of the body and the enzymes listed in Table 10-1, as well as by response to the numerous regulatory neurochemical and hormonal substances produced by the brain, organs of the digestive system, and other tissues that are summarized in Table 10-2. This complex system extracts essential vitamins and minerals from diverse plant and animal foods and breaks down the carbohydrate, protein, and fat molecules in these foods to common subunits that can be absorbed. Once absorbed, these subunits are used as building blocks for the molecules that make up body tissues or for energy production. The structure and function of the digestive system, its response to disease, and the relationship between gastrointestinal function and nutritional status have been reviewed extensively (Floch 1981; Green and Greene 1984; LSRO 1987; Martin, Mayes, and Rodwell 1985; Trowell, Burkitt, and Heaton 1985).

The cellular lining of the digestive tract has a very large surface area and is readily exposed to potential mechanical, thermal, and microbial damage. Its anatomic location, gastric acidity, and elements of the immune system protect it from damage (Cole and Kagnoff 1985; Kagnoff 1983). This topic is reviewed in the chapter on infections and immunity.

Mouth

The physical and chemical breakdown of food begins in the mouth with the physical action of chewing and the enzymatic action of saliva. The parotid and submaxillary salivary glands produce fluids and enzymes that convert smaller carbohydrates and proteins to sugars and amino acids. Fats, however, are not generally altered in the mouth. Secretion of salivary fluid

Table 10-1
Summary of Digestive Processes

Source of Secretion and Stimulus for Secretion	Enzyme	Method of Activation and Optimal Conditions for Activity	Substrate	End Products or Action
Salivary glands: Secrete saliva in reflex response to presence of food in oral cavity.	Salivary amylase	Chloride ion necessary. pH 6.6–6.8.	Starch Glycogen	Maltose plus 1:6 glucosides (oligosaccharides) plus maltotriose.
Lingual glands	Lingual lipase	pH range 2.0–7.5; optimal, 4.0–4.5.	Short-chain primary ester link at *sn*-3	Fatty acids plus 1,2-diacylglycerols.
Stomach glands: Chief cells and parietal cells secrete gastric juice in response to reflex stimulation and action of gastrin.	Pepsin A (fundus) Pepsin B (pylorus)	Pepsinogen converted to active pepsin by HCl. pH 1.0–2.0.	Protein	Peptides.
	Rennin	Calcium necessary for activity. pH 4.0.	Casein of milk	Coagulates milk.
Pancreas: Presence of acid chyme from the stomach activates duodenum to produce (1) secretin, which hormonally stimulates flow of pancreatic juice; (2) cholecystokinin, which stimulates the production of enzymes.	Trypsin	Trypsinogen converted to active trypsin by enterokinase of intestine at pH 5.2–6.0. Autocatalytic at pH 7.9.	Protein Peptides	Polypeptides. Dipeptides.
	Chymotrypsin	Secreted as chymotrypsinogen and converted to active form by trypsin. pH 8.0.	Protein Peptides	Same as trypsin. More coagulating power for milk.
	Elastase	Secreted as proelastase and converted to active form by trypsin.	Protein Peptides	Polypeptides. Dipeptides.

Table 10-1 (continued)

Source of Secretion and Stimulus for Secretion	Enzyme	Method of Activation and Optimal Conditions for Activity	Substrate	End Products or Action
Pancreas (continued)	Carboxypeptidase	Secreted as procarboxypeptidase, activated by trypsin.	Polypeptides at the free carboxyl end of the chain	Lower peptides. Free amino acids.
	Pancreatic amylase	pH 7.1.	Starch Glycogen	Maltose plus 1:6 glucosides (oligosaccharides) plus maltotriose.
	Lipase	Activated by bile salts, phospholipids, colipase. pH 8.0.	Primary ester linkages of triacylglycerol	Fatty acids, monoacylglycerols, diacylglycerols, glycerol.
	Ribonuclease		Ribonucleic acid	Nucleotides.
	Deoxyribonuclease		Deoxyribonucleic acids	Nucleotides.
	Cholesteryl ester hydrolase	Activated by bile salts.	Cholesteryl esters	Free cholesterol plus fatty acids.
	Phospholipase A_2	Secreted as proenzyme, activated by trypsin and Ca^{2+}.	Phospholipids	Fatty acids, lysophospholipids.

Liver and gallbladder: Cholecystokinin, a hormone from the intestinal mucosa—and possibly also gastrin and secretin—stimulate the gallbladder and secretion of bile by the liver.	(Bile salts and alkali)		Fats—also neutralize acid chyme	Fatty acid-bile salt conjugates and finely emulsified neutral fat-bile salt micelles and liposomes.
Small intestine: Secretions of Brunner's glands of the duodenum and glands of Lieberkühn.	Aminopeptidase		Polypeptides at the free amino end of the chain	Lower peptides. Free amino acids.
	Dipeptidases		Dipeptides	Amino acids.
	Sucrase	pH 5.0–7.0.	Sucrose	Fructose, glucose.
	Maltase	pH 5.8–6.2.	Maltose	Glucose.
	Lactase	pH 5.4–6.0.	Lactose	Glucose, galactose.
	Trehalase		Trehalose	Glucose.
	Phosphatase	pH 8.6.	Organic phosphates	Free phosphate.
	Isomaltase or 1:6 glucosidase		1:6 glucosides	Glucose.
	Polynucleotidase		Nucleic acid	Nucleotides.
	Nucleosidases (nucleoside phosphorylases)		Purine or pyrimidine nucleosides	Purine or pyrimidine bases, pentose phosphate.

Source: Murray, R.K.; Granner, D.K.; Mayes, P.A.; and Rodwell, V.W. 1988. *Harper's Biochemistry*, pp. 584–85. 21st ed. San Mateo, CA: Appleton & Lange. Copyright Appleton & Lange 1988, reprinted with permission.

Table 10-2
Gastrointestinal Hormones

Hormone	Localization	Major Action
Gastrin	Gastrin antrum, duodenum	Gastric acid and pepsin secretion
Cholecystokinin (CCK)	Duodenum, jejunum	Pancreatic amylase secretion
Secretin	Duodenum, jejunum	Pancreatic bicarbonate secretion
Gastric inhibitory peptide	Small bowel	Enhances glucose-mediated insulin release. Inhibits gastric acid secretion
Vasoactive intestinal polypeptide	Pancreas	Smooth muscle relaxation; stimulates pancreatic bicarbonate secretion
Motilin	Small bowel	Initiates interdigestive intestinal motility
Somatostatin	Stomach, duodenum, pancreas	Numerous inhibitory effects
Pancreatic polypeptide	Pancreas	Inhibits pancreatic bicarbonate and protein secretion
Enkephalins	Stomach, duodenum, gallbladder	Opiate-like actions
Substance P	Entire gastrointestinal tract	Uncertain
Bombesin-like immunoreactivity	Stomach, duodenum	Stimulates release of gastrin and CCK
Enteroglucagon	Pancreas, small intestine	Unknown

Source: Adapted from Martin, Mayes, and Rodwell 1985.

depends on neuroendocrine regulation and is affected by the sight, smell, taste, and thought of food. The role of salivary secretions is discussed further in the chapter on dental diseases.

Esophagus

Swallowed food enters the esophagus and travels through it to the stomach. At the esophagogastric junction, the lower esophageal sphincter muscle normally prevents gastric fluids from flowing into the body of the esophagus. Neurohormonal mechanisms control the pressure of this sphincter and, as discussed below, may be affected by digestion of certain foods in the small intestine (Pope 1983).

Stomach

In the stomach, food is broken down into increasingly smaller particles and compounds by mechanical, chemical, and enzymatic means. The acidic gastric secretions contain the enzyme pepsin, which converts proteins into short chains of amino acids, and gastric lipases, which, along with lingual lipase, initiate triglyceride digestion. The movement of the stomach empties liquids into the duodenum continuously while food solids are reduced to the consistency of paste. The selective rates of discharge of substances into the duodenum may be related to their caloric density (Brener, Hendrix, and McHugh 1983), to effects of neuropeptides (Morley 1982), or other hormones that control gastrointestinal function.

Small Intestine

Most digestion and absorption take place in the small intestine, as do modulation of fluid balance, orderly advancement of food residues into the colon, reabsorption of bile salts, and absorption of vitamin B_{12}. The specialized absorbing cells, enterocytes, are distinguished by numerous microvilli that greatly increase the absorptive surface area. This surface epithelium contains enzymes that split carbohydrates and small peptides. These cells also convey nutrients to the circulatory and lymphatic systems, which distribute them to the rest of the body. Digestion is primarily accomplished by enzymes secreted by the pancreas and delivered to the small intestine: amylase, which converts starch to sugar; lipase, which splits triglycerides into fatty acids and monoglycerides; and trypsin and chymotrypsin, which split proteins into amino acids and small peptides. Other substances such as secretin, which is produced by the cells of the duodenum, and pancreatic polypeptide help to control the level of intestinal acidity.

The absorption of sugars, peptides, amino acids, and fatty acids proceeds in the upper part of the small intestine (jejunum), whereas bile salts and vitamin B_{12} are absorbed in the distal portion or the ileum. By the time food residues pass the ileum, most usable food molecules have been digested and absorbed, so that only small amounts of carbohydrates (Levine and Levitt 1981), other macronutrients, and fiber are delivered to the colon.

Liver and Biliary Tract

The liver, the largest body organ, synthesizes proteins, oxidizes fat, regulates the release of glucose from glycogen, and detoxifies drugs, hormones, and other potentially deleterious substances. It also converts cholesterol into bile acids and secretes hepatic bile, which is concentrated in the gallbladder before delivery into the duodenum.

Pancreas

The pancreas has both exocrine and endocrine functions that influence digestion and nutrient metabolism. It secretes hormones such as glucagon and insulin into the blood, and digestive enzymes, principally amylase, lipase, and proteases, into the digestive tract.

Colon

The principal functions of the large intestine are to concentrate, store, and excrete food wastes. The colon contains large numbers of bacteria, which produce enzymes that act on the remaining food residues, fiber, and cells and mucus sloughed from the upper intestinal tract. The products of this bacterial digestion and fermentation include short-chain fatty acids (e.g., propionic, butyric); gases such as carbon dioxide, methane, and hydrogen; and other volatile substances (Cummings 1983). The ascending colon has a thinner muscle wall, a greater luminal volume, and a much larger population of bacteria than does the more muscular descending colon, which slows the movement of feces until they are partially dehydrated, concentrated, and ready to be expelled through the anus.

Key Scientific Issues

- Effects of Dietary Factors on Gastrointestinal Function
- Role of Dietary Factors in Intestinal Disorders
- Role of Dietary Factors in Gallbladder Disease
- Role of Dietary Factors in Other Digestive Disorders

Effects of Dietary Factors on Gastrointestinal Function

The diet must provide sufficient nutrients and energy to synthesize the rapidly renewing cells that line the gastrointestinal tract, the enzymes that digest and transport nutrients across the intestinal wall, and the regulatory neuropeptides and other hormones that control these processes. Digestive function can be seriously disrupted by inadequate nutrition as well as by infections, toxic substances, and chronic disease. The composition of the diet can influence the rate of reproduction of bacteria in the intestine and, thus, can affect nutrient absorption. Dietary components affect the morphology and synthesis of the cells that line the digestive tract as well as fecal composition and elimination.

Malnutrition

Nutritional inadequacies can cause abnormalities of the mucous membranes of the mouth, tongue, and digestive tract. Frequent consequences of starvation and protein-energy malnutrition are an inability to absorb or digest food molecules and decreased pancreatic function (Kerndt et al. 1982). As discussed in the chapter on infections and immunity, cellular immune functions are depressed in malnutrition and starvation. Because the cells that line the digestive tract are renewed every few days, nutrient deficiencies can be reflected in ulceration, hemorrhage, or loss of resistance to micro-organisms that are usually not pathogenic.

Atrophy of the gastric and intestinal mucosa is an especially serious consequence of malnutrition. The microvilli flatten and lose much of their absorptive surface and no longer produce adequate digestive or absorptive enzymes. Food passes undigested and unabsorbed into the colon, where bacterial action induces gas production and the influx of water, inducing diarrhea and further damage to the digestive tract. These effects are most severe in young children and cause a characteristic cycle of malnutrition, infectious disease, malabsorption, and diarrhea that is common throughout the developing world (Chandra 1983; Hamilton 1985; see chapter on infections and immunity). They also occur among patients with chronic diseases that interfere with adequate nutrition, anorexia nervosa, impaired immunologic responses to certain food substances (Floch 1981), or severe microbial infections of the gastrointestinal tract (DuPont 1984).

Once the microvilli are reduced, recovery of gastrointestinal function occurs only slowly, if at all. Because food intake induces diarrhea, enteral and parenteral feeding methods that bypass the digestive tract must be employed along with immediate efforts to prevent dehydration, edema, vitamin and mineral deficiencies, and excessive accumulation of body fat (Roediger 1986).

In various conditions in which the bowel mucosa are compromised by disease, macromolecules that are normally excluded may be absorbed. These, especially protein molecules, may alter systemic immune functions (see chapter on infections and immunity).

Effects of Fiber

Most studies of the relationship of diet to gastrointestinal function have focused on the role of dietary fiber. Physiologic responses to dietary fiber occur within the entire length of the gastrointestinal tract. Dietary fibers from different plant sources have diverse chemical constituents; some are soluble and some are insoluble. The major constituents of dietary fiber are cellulose, hemicelluloses, pectins, mucilages, gums, algal polysaccharides, and lignin. Fibers from different foods have different effects on water-holding capacity, viscosity, ion-exchange capacity, binding of minerals and organic compounds, bacterial fermentation, and transit time. Food processing can alter these effects. In general, dietary fibers from many sources increase the flow of saliva, improve feelings of satiety, delay digestion and absorption, bind intestinal bile acids, increase the mass of intestinal bacteria, decrease the time stools take to pass through the bowel, and increase stool weights and frequency of elimination (Trowell, Burkitt, and Heaton 1985). Although animal studies demonstrate that fiber intake increases the length of the intestine and causes greater proliferation of mucosal cells, these effects cannot be readily distinguished from those of other dietary factors, and their applicability to humans is uncertain (LSRO 1987).

Some potential adverse effects have also been observed very infrequently with diets high in fiber, including intestinal obstruction (primarily due to gel-forming fiber); interference with absorption of calcium, magnesium, zinc, manganese, and iron; inflammation of the bowel mucosa (with certain gums); and colonic volvulus (Klurfeld 1987).

Role of Dietary Factors in Intestinal Disorders

Diseases of the gastrointestinal tract affect food consumption, digestion, absorption, and excretion. Although one might expect dietary factors to be important in preventing and treating such conditions, research in this area has not been extensive, and present understanding is limited. The dietary factors most frequently associated with gastrointestinal illnesses are alcohol (liver disease and cancer); inadequate fiber (constipation, hemorrhoids, diverticular disease, and possibly some types of cancer); fat (gallbladder disease and possibly some types of cancer); and substances such as gluten in wheat (celiac disease in genetically predisposed individuals).

Cancer

The effects of dietary risk factors such as alcohol, fat, and food mutagens and carcinogens on the causation of cancers of the gastrointestinal tract, and the effects of substances such as fiber or vitamin A in their prevention, are reviewed in the chapter on cancer.

Celiac Disease

This genetic-immunologic disorder, also known as nontropical sprue or gluten-induced enteropathy, results from an immunologic reaction to the gluten fraction of proteins from wheat, rye, or oats (Chandra and Sahni 1981). Its symptoms may be silent and its prevalence is uncertain, but it is thought to affect about 1 in 2,500 persons in the United States (Gluten Intolerance Group 1982). When patients with this disorder ingest gluten, the cells that line the small intestine undergo atrophy, causing malnutrition, stunting of growth, and anemia. Although strict removal of gluten from the diet alleviates symptoms and restores the integrity of the intestinal mucosa, some immunologic abnormalities may persist. Neither the fundamental defect nor the genetic basis of celiac disease is understood (Cole and Kagnoff 1985).

Constipation

The National Center for Health Statistics has reported that more than 20 of every 1,000 persons surveyed state that they suffer from frequent constipation (NCHS 1986). Although constipation, defined as three or fewer bowel movements per week, can be caused by diabetes, hypothyroidism, uremia, neurogenic bowel disorders, abnormalities in the structure of the colon, rectum, or anus, and by various medications, most constipation cannot be attributed to an underlying disease. Instead, dietary intake patterns are widely presumed to cause this condition, in particular, inadequate consumption of fiber and, especially in the older person, insufficient fluid intake. The effect may vary with coarseness of bran or degree of cooking (Klurfeld 1987), but numerous studies have demonstrated that increased intake of wheat bran and other sources of insoluble fiber prevents constipation and relieves its symptoms (LSRO 1987).

Diverticular Disease

Diverticulosis occurs when diverticula, abnormal outpocketings of the intestinal wall, form in the colon and cause pain in the left lower abdomen without fever. Although diverticula may occur over extensive areas of the colon, they do not usually produce demonstrable muscle thickening, changes in intraluminal pressures, or other noticeable symptoms (Fleischner, Ming, and Henken 1964; Weinreich and Andersen 1976). A closely related disease, diverticulitis, which occurs when the outpocketings become infected, causes constipation and diarrhea, flatulence, abdominal pain, fever, and mucus and blood in the stools (Almy and Howell 1980).

Some experts believe that diverticula occur as a result of increased colonic intraluminal pressure needed to eliminate small, hard stools that form as a result of low-fiber diets (Burkitt, Walker, and Painter 1974). The idea that diverticular disease might result from inadequate intake of dietary fiber is supported by animal studies (Cello 1981); by measurements in humans of intestinal transit times, bowel motility, stool weights, and intraluminal pressures (Burkitt, Walker, and Painter 1974; Painter 1985); and by international comparisons of fiber intake and disease prevalence rates (Mendeloff 1986). Numerous dietary intervention trials have reported beneficial effects of bran and other fiber sources on pain, constipation, and other symptoms as well as on intraluminal pressures (Painter 1985; LSRO 1987). Despite concerns that these studies have not always employed adequate control groups, and despite the needs for further research to define the role of other nutrients such as fat (Manousos et al. 1985) and to identify the most effective sources and types of fiber, fiber supplements are now often used successfully in clinical management of uncomplicated diverticular disease (LSRO 1987).

Inflammatory Bowel Disease

Nonspecific inflammatory bowel disease includes two diseases of the digestive tract: (1) ulcerative colitis, characterized by rectal bleeding, diarrhea, abdominal cramping and pain, loss of appetite, and weight loss, and (2) Crohn's disease, a chronic inflammation anywhere throughout the length of the digestive tract that may induce similar symptoms along with fistulas and narrowing of the bowel (Kirsner and Shorter 1982). Their etiology and pathogenesis is unknown. Patients with these conditions can become severely malnourished. Active cases are usually treated with low-residue diets and caloric supplements (Harries et al. 1983), elemental enteral formulas (Neidich, Schussel, and Sharp 1985), or total parenteral nutrition with complete bowel rest (Ostro, Greenberg, and Jeejeebhoy 1985).

Potential food allergens, such as carrageenan thickeners and cow milk, and low-fiber diets have been suggested as possible dietary factors aggravating these diseases, but evidence to support such inferences is limited (Kirsner and Shorter 1982). Studies of the relationship of low-fiber diets to etiology or treatment of Crohn's disease (Jones et al. 1985) have yielded equivocal results, and the role of diet in inflammatory bowel disease is uncertain at this time (LSRO 1987).

Irritable Bowel Syndrome

This condition of pain, abdominal distension, and alteration in bowel habits is thought to be due to an inappropriate reaction of the intestinal wall to stress (Eastwood and Passmore 1983), motility disturbances, diet (Harvey 1985), or food hypersensitivity reactions (Bentley, Pearson, and Rix 1983). Dietary fiber has been used to treat irritable bowel syndrome with demonstrable improvements in constipation (Fielding 1985; Harvey 1985), but its effects on other symptoms have been equivocal (LSRO 1987).

Lactose Intolerance

An insufficiency of lactase, the enzyme responsible for breakdown of lactose (milk sugar) in the small intestine, can cause lactose intolerance, characterized by abdominal discomfort, pain, and diarrhea as a result of bacterial action on undigested lactose in the colon (Newcomer and McGill 1984). Lactose intolerance is not an inevitable consequence of lactase deficiency. Many lactase-deficient individuals can consume modest amounts of lactose-containing foods with little difficulty. Modification of milk and milk products by addition of lactase, or the use of fermented products such as cheese or yogurt, permits consumption of milk products by such individuals with minimal symptoms (Kolars et al. 1984; Barillas and Solomons 1987).

Genetic absence of lactase beyond the age of 5 or 6 occurs among remarkably high proportions of Asians (85 to 95 percent), Africans (50 to 99 percent), American Indians (85 to 95 percent), and American blacks (70 to 75 percent), as well as among a significant percentage of healthy Caucasians (Gray 1983). Acquired deficiencies can occur as a result of malnutrition (Kerndt et al. 1982) or disease. Reports that lactase activity is lost with aging have not been confirmed (Rosenberg and Bowman 1984).

Role of Dietary Factors in Gallbladder Disease

Cholesterol, precipitated from supersaturated bile, is the principal component of most gallstones in patients from industrialized countries. Dietary and diet-related risk factors for this condition include diabetes, obesity (Diehl et al. 1987), and excess intake of calories and dietary fat (Heaton 1985). Many of these are also risk factors for coronary heart disease.

Low-fiber diets are associated with gallstone formation. In primates, the action of fiber and other substances that bind cholesterol in the intestine is thought to stimulate the liver to increase production of bile acids, thereby increasing cholesterol solubility (Strasberg, Petrunka, and Ilson 1976). Although cellulose, hemicellulose, lignin, and other insoluble fiber components have little effect on blood cholesterol levels, soluble components such as pectin and guar appear to reduce cholesterol levels by 10 to 15 percent (LSRO 1987). Dietary fiber has also been shown to increase the pool of bile acids in laboratory animals (Usuga et al. 1976).

In humans, large doses of wheat bran have been reported to increase bile cholesterol solubility (Pomare et al. 1976). One study has indicated that a fiber-rich diet decreases the cholesterol saturation index of bile significantly (Thornton et al. 1983). Human epidemiologic investigations, however, have not been able to distinguish associations of gallstone formation with varying levels of fiber from associations with other dietary factors such as sugar, alcohol, or other macronutrients (Smith and Gee 1979; Scragg, McMichael, and Baghurst 1984).

Role of Dietary Factors in Other Disorders of the Digestive System

Cirrhosis

The only digestive system disorder besides cancers in the 10 leading causes of death for Americans is cirrhosis (with other chronic liver diseases), and its most powerful dietary correlate is alcohol consumption. This issue is addressed in the chapter on alcohol.

Appendicitis

Studies based on epidemiologic comparisons between industrialized nations and less developed regions have associated low-fiber diets with increased prevalence of appendicitis (Segal 1985; Walker and Burkitt 1985). However, Western trends in fiber intake are not consistent with the decline in appendicitis rates during the past few decades, and not all studies have shown that patients with appendicitis consume less fiber than control subjects (Cove-Smith and Langman 1975). Nondietary factors may be more important (Barker et al. 1986). Nevertheless, reports that children 7 to 18 years of age whose fiber intake is in the upper 50th percentile have a 50 percent lower risk of appendicitis (Brender et al. 1985) warrant further investigation.

Reflux Esophagitis

This disorder, an inflammation of the lower esophagus (heartburn) caused by the backflow of stomach acids, can occur when the lower esophageal sphincter does not contract properly. Alcohol, dietary fat, and both regular and decaffeinated coffee (Cohen 1980; Feldman et al. 1981) have been demonstrated to reduce sphincter pressure and to increase reflux. Spices and tomato and orange juices also may affect some persons.

Ulcers

Gastric and duodenal ulcers are local erosions of the mucosa that result from excessive production of gastric acid and pepsin or from decreased mucosal resistance to these substances. They may result from defects in control of secretion and motility or in synthesis of prostaglandins that either inhibit gastric acid secretion or promote secretion of bicarbonate (Johannson and Bergstrom 1982). The role of nutritional factors in the etiology of ulcers is uncertain. Speculation that refined foods reduce the buffering capacity of stomach secretions (Cleave 1975) has not been confirmed, nor is evidence sufficient to define a causal relationship for linoleic acid, prostaglandins, or peppers in peptic ulcers. The geographic distribution of duodenal ulcers is not consistently associated with fiber consumption (Tovey 1985), nor have clinical studies reported consistent effects of fiber on treatment (LSRO 1987). The observation that increased dietary fiber intake reduces rates of recurrence (Rydning et al. 1982) requires further confirmation.

The bland milk-and-cream-based Sippy diet, used in former years, is no longer recommended as treatment; it has not been demonstrated to improve symptoms better than any other method, is atherogenic, and is deficient in essential vitamins and minerals (Zucker and Clayman 1983). Today, ulcer patients typically are encouraged to consume a varied and balanced diet, taken slowly in four or five small meals a day, but limited in alcohol, coffee, and other substances that lead to discomfort and pain. Patients should avoid late evening snacking that stimulates nocturnal acid secretion (Floch 1981). Patients are also advised to avoid cigarette smoking, which accentuates symptoms and retards healing, and aspirin, which irritates the gastrointestinal mucosa and can cause mucosal hemorrhages. It should be noted that there are many current, very effective pharmacologic therapies for the management of ulcer disease.

Implications for Public Health Policy

Dietary Guidance

General Public

Dietary fat, fiber, and alcohol are significant factors associated with gastrointestinal diseases, although the great variety of these conditions makes generalizations difficult. Because diets that contain a large proportion of calories from fat may be low in fiber, it is often difficult to separate the effects of these substances on gastrointestinal disease. Thus, current evidence on whether dietary fiber helps prevent diverticulosis is not conclusive. Similarly, whether dietary fiber helps prevent inflammatory or irritable bowel disease is uncertain. Nevertheless, evidence that dietary fiber helps treat and prevent constipation and manage chronic diverticular disease suggests the prudence of consuming diets higher in fiber and lower in fat.

The strong cause-and-effect association between excessive alcohol consumption and the development of chronic liver disease and cirrhosis (as reviewed in the chapter on alcohol) emphasizes that persons who consume alcoholic beverages should do so in moderation. Epidemiologic associations between diet and some types of gastrointestinal cancer (as reviewed in the chapter on cancer) suggest—but do not yet prove—that consuming less fat and alcohol and more fiber would help reduce the risk for these cancers.

Evidence on the role of dietary factors in the development of gastric or duodenal ulcers or reflux esophagitis is insufficient to make recommendations at this time.

Special Populations

Higher intakes of dietary fiber can prevent or relieve symptoms of constipation and chronic diverticular disease. Qualified health professionals should inform persons with these conditions about foods with relatively high fiber contents. Individuals with celiac disease should be provided with information on foods free of wheat gluten. Those with inflammatory bowel disease, irritable bowel syndrome, lactose intolerance, gallbladder disease, heartburn, and ulcers should be provided with guidance on diets appropriate to their conditions.

Nutrition Programs and Services

Food Labels

Evidence related to the role of dietary factors in gastrointestinal disease suggests that food manufacturers should include on package labels information about nutritional content of the food, especially for fat and carbohydrate components (and including fiber components to the extent permitted by analytical methods).

Food Services

Evidence related to the role of dietary factors in gastrointestinal diseases suggests that food services should include provisions for adequate intake of high-fiber and low-fat foods.

Food Products

Evidence related to the role of dietary factors in gastrointestinal diseases suggests that the public would benefit from additional products that are low in fat and calories and higher in fiber.

Special Populations

Persons with gastrointestinal diseases should receive counseling and assistance in developing appropriate diets for their particular condition. Qualified health professionals should provide appropriate training and enteral or parenteral nutritional support to persons with conditions that prevent food ingestion, cause malabsorption, or impair bowel function.

Research and Surveillance

Research and surveillance issues of special priority related to dietary factors affecting gastrointestinal function and diseases of the gastrointestinal tract should include investigations into:

- The prevalence of gastrointestinal diseases among the population.
- The influence of dietary factors such as specific dietary fibers, fat, and calories on development and function of the digestive tract.
- The influence of dietary factors on the development and release of enzymes and hormones that affect gastrointestinal function.
- The role of intestinal flora on nutrient bioavailability.
- The most effective nutrient-related interventions to improve the recovery of intestinal function following episodes of malnutrition or disease.
- The mechanisms by which dietary fiber may work in the prevention and treatment of bowel cancer, appendicitis, diverticular disease, gallbladder disease, and other gastrointestinal conditions.
- The identification of specific dietary factors that might influence the causation, prevention, and treatment of celiac disease, inflammatory and irritable bowel syndromes, ulcers, and other gastrointestinal disorders.
- The most effective means to achieve dietary counseling to help alleviate gastrointestinal disorders.

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